should be removed and tracheotomy performed, if suffocation is threatening.

Owing to the density and spreading of scleroma in Europe, H. von Schroetter and Streitt agitate of late that these cases should be officially reported and always watched officially, since the disease spreads more and more and invades districts which have been heretofore decidedly free from scleroma.

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ACUTE PERITONEAL INFECTION AND ITS RELATION TO INTESTINAL OBSTRUCTION.

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THE anatomical features of the peritoneum which have an important bearing upon its inflammations are the following: The surface of the peritoneum is constituted of extremely thin endothelial cells, lying upon a very delicate and rich plexus of lymphatic spaces and capillary bloodvessels. Over the intestines these circulatory channels are so fine that peritoneal cells may almost be said to lie upon the muscular coat of the bowel. The presence or absence of stomata in certain parts of the peritoneum is still undetermined. The phenomena of the transudation of fluids in the peritoneum is sufficiently explained in the laws of osmosis; and the absorption of solid materials has been demonstrated to be accomplished through their inclusion by leukocytes. The absorptive power of the peritoneum, both for fluid and solid particles, is very great. It is needless to say that the different bacteria which cause peritonitis each produce a different sort of inflammation. These differences will not be discussed in this paper. We shall deal only with the ordinary form of acute peritonitis, having as its usual characteristic the presence of the common colon bacillus, and commonly associated in the beginning with other organisms.

A small number of infective bacteria, when implanted upon the peritoneum, become absorbed and disappear; when a large number are present, or when there is already present nutritive material, such as serum, fibrin, or necrotic tissue, the bacteria multiply, and a progressing peritonitis develops. In the first instance there are present all of the phenomena of inflammation: a single bacterium excites a minute local irritation by its mechanical presence and the irritation of its toxins; the underlying capillaries become dilated; a small amount of serum transudes; a leukocyte passes through upon the peritoneal surface, envelops and destroys the bacterium; the source of irritation is eliminated, and the tissues return to their normal state. In the event of the second instance there are present a large number of bacteria, or the growth *in situ* of a large number by virtue of the presence of nutritive material, which not only permits them to multiply, but interposes substance between them and the surface of the peritoneum, thus placing them out of reach of the leukocytes. In this event an amount of toxin is evolved sufficiently large to produce irritation and consequent exudation, which lifts the bacteria off the peritoneum and enables them again to grow beyond the reach of the leukocytes. Thus a vicious circle is established. The more bacteria there are the easier it is for them to multiply. The leukocytes which appear upon the surface of the peritoneum are killed by the ptomains in the fluid which envelop them. They fall from the peritoneum and become pus cells. Such an inflammation spreads and continues to spread, the exuded serum acting as a vehicle to convey the bacteria. Fibrin coagulates upon the surface of the peritoneum. This may be digested and broken down by bacterial products, or it may be exuded from two opposing surfaces of peritoneum, and thus cause their agglutination. This latter is the usual means by which the further progress of peritonitis is checked, and shows that the convection of the bacteria is accomplished by the agency of the fluid which they have caused to be excreted.

In the extremely virulent and rapid cases of peritonitis, death often takes place before any appreciable exudate has developed. In these cases I have observed that the inflammatory process does not travel past pre-existing adhesions, any more than it does in the cases in which the infection is quite clearly conveyed by fluid. The peritoneum is found dull and lustreless, swollen, dark red in color, with here and there some thin patches of fibrin.

However, it is not the surface manifestations, as is generally taught, that most concerns us in peritonitis. It is the inflammation in the structures beneath the peritoneum that is responsible for the high mortality, and that makes peritonitis a dreadful disease.

I shall undertake to show that the absorption of septic material from the so-called peritoneal cavity has little to do with the symptoms of peritonitis, that there is no peculiarity about the peritoneum which renders its inflammations different from other structures, and that it is not the disease itself which is fatal but its complications, which induce an actual condition of intestinal obstruction.

Let us see. When the peritoneal surface has become infected and an inflammation has developed, besides the surface changes above alluded to there is present an inflammation of the subserous structures as well. That means there is a dilatation of the capillaries, an exudation of white blood cells, and an infiltration with round cells of these underlying structures. These capillaries lie directly upon the muscular coat of the bowel, and it is this and the accompanying nerve filaments that are as much affected by the inflammation as the serosa cells of the surface. In peritonitis more or less of the whole thickness of the muscularis is infiltrated with the products of inflammation. The serosa cells have but little function more than a covering. The absorption and exudation of fluids probably take place in the interstices between them. But the underlying structures have a function of such great importance that the life of the individual depends upon it. I refer to the contractions of the muscularis, which give rise to the peristalsis of the intestine. A muscle fibre infiltrated with the products of inflammation loses its power to function. This is due to the mechanical interference caused by foreign cells, to pressure upon the nerve filaments, and to the toxic effect of the ptomains of inflammation upon the cells of both nerves and muscles. Thus the underlying bowel muscle becomes paralyzed.

Immediately after a segment of bowel becomes paralyzed, the gaseous pressure from within causes it to rise from the coils of intestine to the highest position it can reach. The question of hydrodynamics now enters. The paralyzed loop is always at the top, and the bowel contents must be propelled upward to reach it. If the patient could be reversed, and the paralyzed loop made to occupy a dependent position, then it would be filled with fluid or solid contents, which it must needs propel upward and which would be impossible because of its paralysis.

However, we see in these speculations one of the reasons why patients with peritonitis do better with the upper part of the trunk elevated. The bowel is not able to propel its contents, either fluid or solid, upward and through the retained gas in the paralyzed loop to catch the incline downward on the distal side. An effort at propelling material into the paralyzed segment at the top ends in increasing its internal pressure and further distending it; and when the effort at propulsion has ceased, the internal pressure being somewhat relieved, the diseased bowel slightly contracts, the gas expands, and the contents under propulsion are driven back again. Thus an actual state of intestinal obstruction exists.

As to the theoretical acceptability of these statements, we know the end facts to be clinically verified.

Clinically, paralysis of the bowel is the same thing as intestinal obstruction. It gives rise to the same symptoms. A segment of intestine completely filled with material which it has not the power to propel onward is the seat of intestinal obstruction, it matters not whether this material is cancer, a foreign body, fluid, or gas.

The clinical pictures are so similar that were it not for the peculiar symptom of rigidity of the abdominal muscles in peritonitis it is often difficult or impossible to distinguish intestinal obstruction from peritonitis. Later, when the bowel has become distended in intestinal obstruction, it becomes paralyzed, and permits the migration of bacteria from its lumen to the peritoneum. A peritonitis then develops, and often there are present conditions similar to what there would be were the peritonitis the cause and not the result.

In the ordinary case of peritonitis with paralysis of the bowel met with by the surgeon, he sees, upon opening the abdomen, many coils of intestine distended and inflamed. A small part of this inflammation and distention is due to peritonitis: that having taken place causes an intestinal obstruction, which in turn causes a dilatation of the bowel above, with paralysis and transmigration of intestinal bacteria from the mucous membrane to the peritoneum. The peritoneum in this event becomes inflamed. At first it is dry. It is at this stage that it is usually seen by the surgeon. Later secretion is present, and it becomes difficult to discover whether the paralysis of the bowel preceded or followed the peritonitis. In all such cases there are present an infective paralysis below and a paralytic infection above.

The subjective symptoms of these two conditions are about the same, and are due to the same conditions; yet in text-books and teaching a great differentiation is attempted, based upon the erroneous idea that the important feature in peritonitis is the absorption of septic poisons from the peritoneal cavity. The great power of absorption of the peritoneum is cited. But the normal peritoneum is one thing, and the inflamed peritoneum is another. While the area of the peritoneum is equal to the cutaneous area of the same individual, still a peritonitis of this whole area never occurs. There is no such thing as a general peritonitis. Moreover, the septic products of peritonitis are not in contact with the normal and absorbent peritoneum, but with a surface which more closely resembles the lining of an abscess. Inflamed peritoneum is so changed and thickened by the deposit of fibrin and young inflammatory cells, with new and dilated blood channels, that it resembles granulation tissue more than it does peritoneum. These changes in the peritoneum pretty generally precede the pus. But even where pus suddenly bursts upon a normal peritoneal surface, the irritation which it causes quickly produces an inflammation, and the whole texture of the peritoneum is changed. I doubt very much whether the septic absorption from pus in the peritoneum is much greater than that from a simple abscess in any other part of the body.

The fever in peritonitis is low compared with the rapid pulse and other indications of depression. The low temperature is the temperature of the peritonitis; the high pulse rate is the pulse of intestinal obstruction. Often the temperature in purulent peritonitis is but little above normal. This is the case with pus lying free in the peritoneal sac when not restrained under pressure, and it is what would be expected in an abscess in any other part of the body. A simple pool of pus, not under pressure to drive its toxins into the lymphatics and blood channels, does not give much fever; but pus retained under pressure, whether in the thigh or about the appendix, does.

In the worst cases of peritonitis the disease runs a rapid course, the peritoneum is intensely inflamed, a paralysis of much of the intestine rapidly develops, and the patient dies with all the symptoms of intestinal obstruction and with scarcely any fluid in the peritoneal sac. Here the disease is distinctly a disease of the bowel.

In thrombosis and embolism of the mesenteric vessels a complete paralysis of the bowel results from the discontinuance of the blood circulation. Here is a condition which comes under the heading neither of peritonitis nor intestinal obstruction, yet it so closely resembles these that it is commonly mistaken for one or the other. So striking is the similarity to intestinal obstruction that Deckart[1] in his work on thrombosis and embolism of the mesentric vessels gives it the sub-title of "A Contribution to the Study of Ileus."

A perforation of the stomach posteriorly, causing a circumscribed peritonitis, gives an entirely different picture from that of a perforation of the small intestine with the same area of peritonitis. The mere matter of septic absorption from the peritoneum is here shown to be of little importance, notwithstanding the much talked of absorbent power of the diaphragmatic peritoneum. The case with the perforation of the intestine dies with the symptoms of intestinal obstruction. The stomach case, if the bowel is not paralyzed, presents no more symptoms of sepsis than it would if the same pus were confined in an abscess of the lumbar muscles.

Puerperal peritonitis is a particularly fatal disease, and it is often cited as an example of the serious character of peritonitis. The patient often dies before a paralysis of the bowel has had time to develop. The symptoms are those of profound septic intoxication. Here the peritonitis is not the chief thing, as is generally taught. The infection is due to a streptococcus of great virulence, and while the peritoneum is involved, there is also an involvement of the uterus and the rich lymphatic structures of the broad ligament and the pelvic spaces, and in many cases the patient dies before peritonitis has begun. There are other cases without symptoms of intestinal obstruction or meteorism. These are often cases with empty intestines, or cases in which the gas-producing organisms in the intestines are few, or cases in which the infecting organism was not capable of penetrating the intestinal wall. Such cases are very rare.

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[1] Grenzgeb. d. Med. u. Chir., 1900, vol. v.

When we come to consider the morbid phenomena of acute intestinal obstruction, we discover that the symptoms of this condition are not alone due to the simple interruption of the fecal current. This alone does not supply a satisfactory explanation. These symptoms are complex, and depend upon the absorption of stercoral products, upon circulatory disturbances in the intestinal wall and mesentery, and upon disturbances of the nerve functions in these parts. Aside from the pathological changes which show the ordinary fatal case of acute peritoneal infection to be a disease properly belonging to the intestinal obstruction group, and not to the septic infective diseases, the clinical picture alone would place it in this category. In both alike are found the rapid pulse, the comparative low temperature, meteorism, interruption of the fecal current, fecal vomiting, the pinched expression, the sunken eyes, cyanosis, the weak heart, and the symptoms of profound shock. Septic absorption from the peritoneum adds but little or nothing to this clinical picture.

A STUDY OF THE MESENTERIC GLANDS IN THEIR RELATIONS TO TUBERCULOSIS.

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This investigation involved a study of the mesenteric glands in 70 cases, divided into two principal groups, the tuberculous and the non-tuberculous. The tuberculous group numbered 49, and included those in which a distinct tuberculous focus or foci, great or small in extent, could be demonstrated somewhere in the body. The non-tuberculous group numbered 21, and included those in which it was impossible by gross examination to demonstrate in any part of the body a tuberculous lesion.

The mesenteric glands were placed under three headings: (1) the apparently nomal, (2) the enlarged, and (3) the tuberculous. The normal glands need no comment. Under the enlarged glands (a rather indefinite term) were included all those in which no